Effectiveness of Electroconvulsive Therapy in Persistent Methamphetamine Psychosis: A Pilot Study

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Original Article

Abstract

Background: Persistent methamphetamine (METH) psychosis is a psychotic state beyond 1-month after abstinence, for which there is no effective treatment. This study aimed to evaluate the effectiveness of electroconvulsive therapy (ECT) in persistent METH psychosis patients hospitalized at Shahid Beheshti Hospital, Kerman, Iran, from 6 September 2012 until 6 September 2013, who were not remitted after treatment with olanzapine.

Methods: This research was a pilot study on hospitalized patients. After 4 weeks of treatment with olanzapine, 10 out of 71 studied patients did not show complete remission of psychotic symptoms despite their response to the treatment. The mentioned 10 patients were divided into 2 groups by random digit numbers. 5 patients had continued olanzapine and other 5 received 6 sessions of bilateral ECT every other day in addition to olanzapine.

Findings: Remission rate of patients in the initial 4 weeks was 78.7%. Reduction in total brief psychiatric rating scale (BPRS) scale at the end of 1-week compared with the next week demonstrated improvement in the symptoms until the end of the study. There was no significant difference in BPRS scores between weeks 4 and 6 in the two groups.

Conclusion: This research demonstrated that few sessions of ECT in persistent METH psychosis will not lead to remission in all patients.

Keywords: Methamphetamine psychosis, Treatment, Electroconvulsive therapy

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Introduction

Illicit methamphetamine (METH) abuse is a serious health and social concern in many countries worldwide, including Iran. In Iran, a transition is happening from traditional patterns of using opium and opium residue to METH abuse,[1] which was rare in the past decades[2] but is significantly growing in the recent years,[1,3] especially among the youth.[4] Some pieces of evidence are available for the rapid increase of METH abuse in Iran since 2008, the substantial portion of which is originated from small laboratories.[5] Although no definite estimation exists about the prevalence and incidence of METH abuse in Iran, some reports have identified it as the second most prevalent substance in the country.[5]

Amphetamines are widely prescribed for conditions such as attention deficit hyperactivity disorder with very little risk of psychosis.[6,7] In contrast, illicit use of METH, especially in supratherapeutic doses taken by the non-oral routes of administration can cause psychotic symptoms more frequently.[8]

Persistent METH psychosis (beyond 1-month after abstinence) has been extensively observed in Japan for more than 50 years; however, it is rarely discussed in the American literature, possibly because some of such cases are misdiagnosed in the United States as primary psychotic disorders. This issue is reflected in the diagnostic lexicon of Diagnostic and Statistical Manual of Mental Disorders-4th Edition (DSM-IV), which defines substance-induced psychosis to be persistent for 1-month or less after the last substance use.[9] Until recently, Japanese substance abusers have rarely consumed drugs other than METH; thus, investigators could assess the effects of METH without the confounding effects of other substances.[10] Considering the growing public health problem of METH abuse in many countries in the world, the distinction between persistent METH psychosis and a primary psychotic disorder has become increasingly important.[11]

As mentioned above, most of the voluminous international literature on METH psychosis has occurred in Japan and Japanese studies have frequently reported discouraging results with standard antipsychotic drugs.[12] One case report of electroconvulsive therapy (ECT) in METH psychosis treatment is entitled "remission of persistent METH-induced psychosis after ECT;" however, to the best knowledge of the present authors, no other studies have been conducted in this regard thus far; only one work has demonstrated the effects of repeated electroconvulsive shock (ECS) on METH-induced behavioral abnormalities in mice.[13] ECT is considered for the treatment of refractory psychosis regardless of the diagnosis and has been shown to be effective among the patients with refractory psychotic symptoms associated with major depression, bipolar disorder, Huntington chorea, Parkinson's disease, and vascular depression among other disorders.[14]

In this center, ECT was being used in the treatment of METH psychosis. According to the above reasons, this study was done to evaluate the effectiveness of ECT in persistent METH psychosis. As a result, clinicians must be alert to the possibility of persistent METH psychosis and wish to consider ECT in refractory cases; thus, after remission, the patients would return to their full baseline level of social and occupational functioning.

Persistent METH psychosis is defined as a psychotic state beyond 1-month after abstinence, for which there is no effective treatment. The present work aimed to evaluate the efficacy of ECT in METH psychosis patients, who were not remitted after treatment with olanzapine.

A recent Australian study which was done on 277 non-treatment-seeking illicit METH users with no prior diagnosis of schizophrenia or other psychotic disorders demonstrated that 51 (18.0%) had "clinically significant" psychotic symptoms.[8] Similarly, a recent U.S. work on 42 cocaine-dependent and 43 METH-dependent individuals, which aimed to exclude patients with other axis I disorders, reported psychotic symptoms of at least some type among at least 60.0% of both groups.[12] Although recent studies have noted the limitations in definition and diagnosis and the variety of studied populations, they have shown that between 26.0 and 46.0% of people with METH dependence have psychotic symptoms.[9] Smoking and injection are the main routes of METH abuse;[5] it seems that smoking is the most common route of METH abuse in Iran.

Multiple studies in Japanese,[15] Taiwanese,[16] Australian,[17] and Thai[10] populations have reported high frequency of persecutory delusions

and auditory hallucinations among people with METH psychosis. Delusions of reference, visual hallucinations, and thought broadcasting are other frequently reported symptoms.[10,15,16] In a study on 149 METH abusers, mean latency of the first use of METH to the onset of psychosis was 5.2 years; another work compared METH injectors and smokers and observed the latency of 4.4 years in injectors and 1.7 years for smokers.[11]

Although psychotic symptoms usually remit after acute intoxication, some patients have exhibited prolonged psychosis for weeks or months after stopping METH.[9] In a Japanese study, 52.0% of 104 hospitalized METH users with METH psychosis psychotic symptoms abated within 1-week; in contrast, 26.0% had symptoms that persisted for more than 1-month and 16.0% of their symptoms persisted for more than 3 months of abstinence.[18] In a Taiwanese work, which was performed on 174 hospitalized METH users with METH psychosis, 17.0% of symptoms persisted for more than 1-month of abstinence.[16]

Japanese investigators have mentioned three types of METH psychosis based on the time course after abstinence: Transient type with brief duration (duration < 1-week), prolonged type in which psychotic state continues up to 1-month of last METH use, and persistent type that is represented with psychotic state beyond 1-month after abstinence.[15] Japanese researchers have estimated that 40.0% of various types of METH psychosis suffer from the persistent type.[15,17] There are few works on METH psychosis treatment. One early Japanese study described 74 patients with METH psychosis observed prior to the era of neuroleptics, in which about two-third of the patients remitted within 20 days after stopping METH, but about 10.0% displayed psychosis, which lasted for more than 6 months and in some cases even a few years.[19]

In another early study, Teraoka reported "schizophrenia-like symptoms" in 32 (28.0%) out of 114 former METH users who were followed up after 8-12 years.[20] A more recent investigation described 132 consecutive patients with METH psychosis who admitted to a Tokyo hospital from 1978-1987; about 28.0% of them required more than 61 days of hospitalization.[21] A subsequent report from the same group described 104 additional patients with METH psychosis admitted to the same hospital from 1988-1991; despite abstinence from METH and administration of antipsychotic drugs, 16 (15.0%) required more than 3 months of hospitalization.[18] A more recent Japanese work in another center observed that 28.0% of inpatients with METH psychosis had symptoms that persisted for longer than 6 months of abstinence.[18]

Similarly, Akiyama mentioned 32 female prisoners treated with standard antipsychotic medications for METH psychosis, none of whom were reported to have displayed psychosis or a diagnosis of schizophrenia prior to METH use. As prisoners, the women had no access to further METH. Although the exact number of those who did and did not respond to the treatment could not be calculated from the article, it appears that a majority of patients, especially those with more severe symptoms, were still symptomatic even after many months of antipsychotic treatment.[15]

Paucity of research in the area of treatment of METH psychosis was highlighted by a 2008 Cochrane Review[22] that found only one randomized controlled trial of the treatment of amphetamine psychosis in the related literature, which was a 4 weeks investigation on 58 patients with amphetamine psychosis in Thailand.[23] There was no significant difference in terms of clinical efficacy between olanzapine and haloperidol and 27 (93.0%) out of 29 patients on olanzapine and 23 (79.0%) out of 29 patients on haloperidol had at least 40.0% improvement on brief psychiatric rating scale (BPRS) after 4 weeks; however, it was not clear how many patients still exhibited psychotic symptoms at the endpoint. Furthermore, because of the lack of placebo arm, it is difficult to judge if improvement is the natural course of amphetamine psychosis alone or not. Two recent case reports have separately found olanzapine[24] and risperidone[25] to be effective for METH psychosis. Because ECT has not been widely used in Japanese psychiatry, Japanese literature lacks ECT trials of METH psychosis.[9]

Methods

The present study was designed as a randomize control trial with 42 sample size out of 105 METH psychosis. It was performed on hospitalized patients from September 6, 2012 to September 6, 2013 at Shahid Beheshti Hospital, Kerman, Iran. It was supported by Research Center for Science

and Neurology under the ethical code of K/93/95.

For collecting the sample size, the study with olanzapine was started; the reason for choosing olanzapine as the anti-psychosis drug was related to the only randomized controlled trial research conducted in Thailand, in which 93.0% of patients showed at least 40.0% reduced symptoms based on BPRS.[23]

After admission of the patients who were diagnosed to have METH psychosis confirmed using positive METH test, a DSM-IV-based semi-structured interview was conducted to confirm the initial diagnosis; then, exclusion criteria were reviewed: Positive family history of mood or psychotic disorders (e.g. bipolar and schizophrenia) among first and second degree relatives, physical diseases such as diabetes and epilepsy requiring medical treatment, and being depended or abusing other substances including cannabis and benzodiazepines, but not opium, because most patients were opium consumers and the sample of this study would not be collected during the anticipated duration (1-year).

After 1-year from the beginning of the study, 71 patients entered the study and, out of these 71 patients, 24 patients left the study. Out of 47 (instead of 105) patients who were remained and received 4 weeks of treatment with olanzapine, 37 patients were recovered and 10 psychotic (instead of 42) patients were remained for the trial (based on the score of higher than 4 in each of psychotic subgroup items of BPRS questionnaire). The patients were assigned to two groups using random digit numbers; in the first group, drug prescription was continued and, in the second group, 6 sessions of bilateral ECT were added to the drug intake every other day for 2 weeks (Figure 1). Maximum length of hospitalization was 6 weeks and BPRS questionnaire was filled for 7 times to assess the patients' symptoms (at baseline and then once per week until the end of week 6).

Why the sample size was small in this study? Two responses can be mentioned as follows:

- In this work, persistent METH psychosis was approximately 21.0% [i.e. half of what Japanese investigators reported (40.0%)]
- Another reason for the small sample size was 34.0% dropout, 20.0% (n = 14) of which was due to early discharge of patients by their families because of either partial and/or lack of response to the treatment that can be a confounding factor. Moreover, in 3.0% (n = 2) of the cases, the physician started ECT earlier than 1-month from the start of the treatment due to their intensity of symptoms. 8.0% (n = 6) of the data loss was related to lack of coordination of the treatment team and the point that more than one physician visited the patients at the education hospital. It seems that better declare of information before the study could reduce this 8.0% of dropout. The remaining 3.0% (n = 2) of data loss was because of drug reaction (delirious state).
- Due to the small sample size, the investigation was considered to be a pilot study. It is well-known that pilot studies are a necessary first step in exploring novel interventions and novel applications of interventions and their purpose is to examine the feasibility of an approach and point out the modifications required for planning and designing a larger efficacy trial. Pilot studies can be used in all types of studies such as randomized control trials.[26]
- Demographic variables of the patients (age, sex, marital status, educational level, occupation, and onset of METH abuse) were compared between the two groups (Table 1). BPRS which gives ratings of 1 (absent) to 7 (extremely severe) for each item was filled to evaluate the patients' symptoms. This scale has been translated into Persian and was utilized in study of Fallahi.[27] Reliability coefficient of the tool has been determined by Chronbach's alpha as r = 0.80 in some studies. For measuring response to the treatment, the scores of the 18 items were summed and the total score was recorded. Then, the total score from one evaluation was compared with that of the next.[28]

Olanzapine and buprenorphine were administered for the patients with psychotic symptoms and addiction, respectively. Also, antidepressant and mood stabilizers were administered for the patients with severe mood disorders due to METH. Prescribing antidepressants and mood stabilizers can be a confounding factor. To remove its effect, it was better to compare people who received this drug with those who did not. However, such a comparison was not made in this investigation.

Except olanzapine, no other antipsychotic drugs were used. Olanzapine started with 5-10 mg and continued up to the maximum 20 mg as rapidly

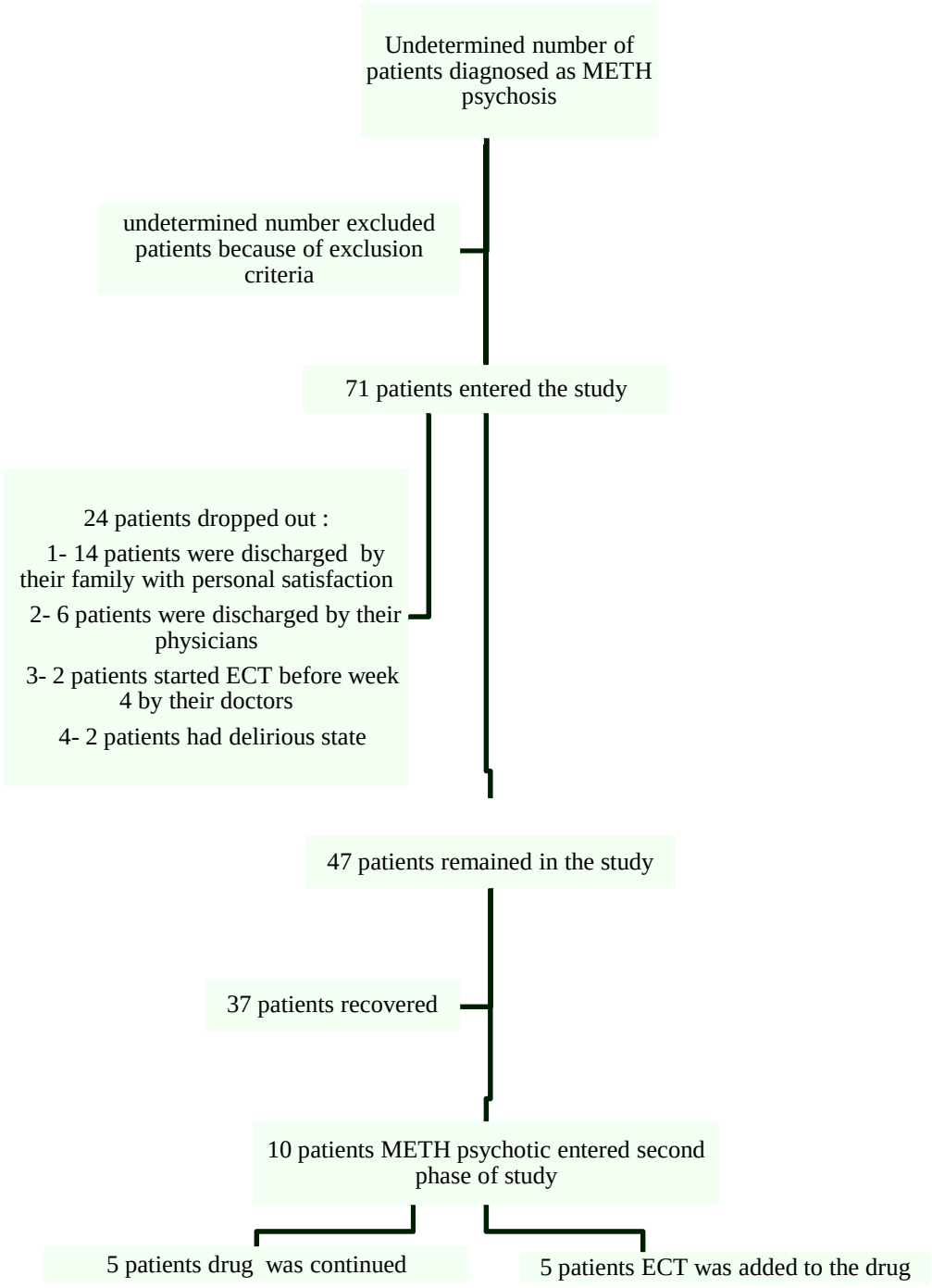

Figure 1. Organizational chart showing the patients' entrance into the study
METH: Methamphetamine; ECT: Electroconvulsive therapy

as possible in 1-week. Finally, all of the patients received 20 mg of olanzapine in both groups.

Although a better response can be observed at higher doses, olanzapine dose was not increased to more than this amount due to increased probability of its complications. It should be mentioned that, the aim of olanzapine treatment was to spend 1-month without taking METH, since according to ethical considerations and hospitalization limitations, the patient with psychotic symptoms could not be hospitalized without any medications.

Categorical and quantitative data were compared between the two groups using chi-square and independent t-test. Two-way repeated-measures ANOVA was also used to compare the course of treatment outcome along the 6 weeks of the study.

Similar to other trials, this study had several limitations: The first and main limitation of the present study was small sample size due to the failure in sample collection and lack of remission after ECT in all the cases (5 patients) of this study was confounded with small sample size. A pilot study is not a hypothesis-testing study and safety, efficacy, and effectiveness are not evaluated in a pilot work. Thus, this pilot study cannot determine the rate of remission after ECT, but offers little insight into lack of remission after ECT in all persistent cases.

Second limitation: It should be considered that some of the patients' symptoms might have been attributable to an underlying primary axis I disorder.

To reduce such likelihood, the patients who had positive family histories of schizophrenia or bipolar disorders were not enrolled the study. In addition, the present patients showed good academic and occupational functioning prior to regular METH smoking. Also, psychotic symptoms appeared after long term use, a pattern which could indicate amphetamine-associated neurotoxic effects. The latency from the first METH smoking to the onset of psychosis was 3.5 years, similar to other studies. In Matsumoto et al.'s study, it was 1.7 years [standard deviation (SD) = 2.0] among smokers and 4.4 years in injectors.[29] Also, it was 5.2 years in the study by Ujike and Sato.[17] Considering the above chronology, illicit METH smoking appears to be the most parsimonious explanation for the etiology of the symptoms.

Moreover, in Iran similar to other countries, there are a considerable number of multi-drug abusers; thus in this study, cannabis and/or benzodiazepine abusers were excluded. However, there is a traditional pattern of using opium and opium residue in Iran which could not be excluded. Because equal number of patients in both groups consumed opium, this issue cannot be a confounding factor, but could raise the following question for the present authors: Whether using opium by these patients plays a role in the remission failure after ECT or not.

Results

As depicted in table 1, demographic characteristics were not significantly different between the two groups. Mean age ± SD for the members of drug group was 36.6 ± 4.9 years, while it was 39.4 ± 11 years for drug + ECT group (P = 0.618). Duration of using METH for the members of drug group was 43.2 ± 24.9 months, while it was 40.8 ± 10.7 months for drug + ECT group (P = 0.848). Number of patients with part-time jobs (% within group) was 2 (40.0%) and the unemployed was 3 (60.0%) in drug group, whereas they were 1 (20.0%) and 4 (80.0%) in drug + ECT group, respectively (P > 0.050). In terms of education, 2 (40.0%) were below high school diploma and 3 (60.0%) had high school diploma in drug group, while they were 1 (20.0%) and 4 (80.0%) in drug + ECT group, respectively (P > 0.050).

Figure 2 indicates that there was no statistically significant difference in response to treatment between the two groups that is demonstrated with non-significant P = 0.167 for between-subject effect and 0.784 for interaction. P-value for within-subject effect was significant (< 0.001). There was no significant difference between mean BPRS score at the end of weeks 4 (37.4 ± 13.1 vs. 36.0 ± 9.7) (P = 0.852) and 6 (30.8 ± 5.7 vs. 30.2 ± 7.4) (P = 0.890) in one group with the same scores in the other group. In other

Table 1. Comparing demographic variables in both groups

Demographic variables		Drug	Drug + ECT	P
Age (year) (mean ± SD)		36.6 ± 4.9	39.0 ± 11.0	0.618
Duration of consumption (month) (mean ± SD)		43.2 ± 24.9	40.8 ± 10.7	0.848
Job (within group) [n (%)]	Unemployed	3 (60)	4 (80)	> 0.050
	Part time	2 (40)	1 (20)	
Education (within group) [n (%)]	Under diploma	2 (40)	1 (20)	> 0.050
	Under diploma	3 (60)	4 (80)	

SD: Standard deviation; ECT: Electroconvulsive therapy

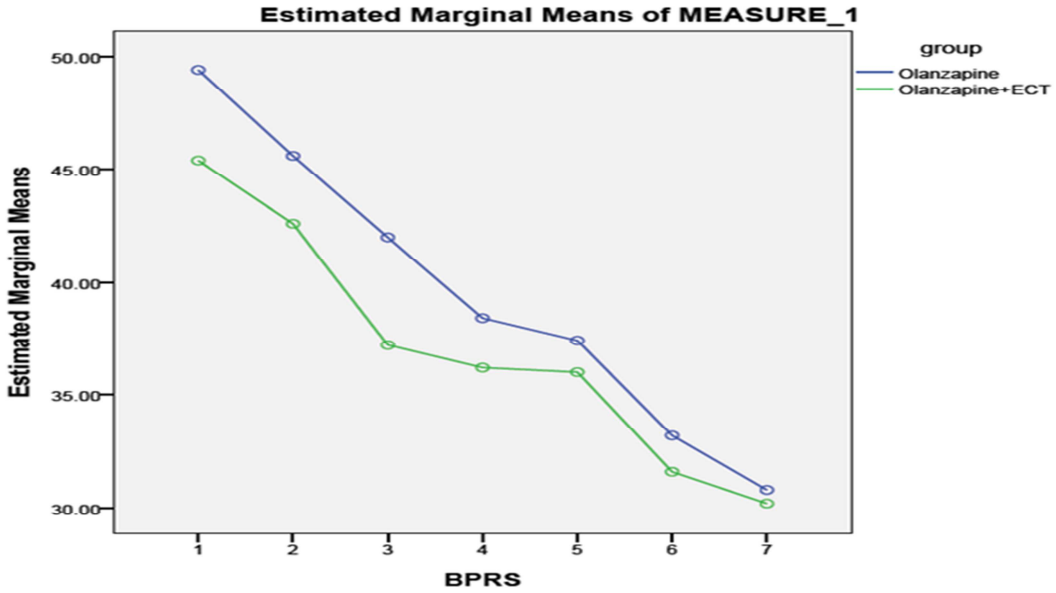

Figure 2. BPRS (brief psychiatric rating scale) scores of 6 weeks of treatment in two groups
ECT: Electroconvulsive therapy; BPRS: Brief psychiatric rating scale

words, in this preliminary clinical trial, remission was not observed for ECT. In the present work, mean latency from the first METH abuse to the onset of psychosis was about 3.5 years.

Discussion

To the best knowledge of the present authors, the current pilot study was the first work that evaluated ECT in persistent METH psychosis (psychosis which is prolonged to more than 1 month after abstinence). In none of the 5 studied patients treated with olanzapine and none of the 5 patients treated with olanzapine plus 6 sessions of bilateral ECT, remission was observed. There was no statistically significant difference in response to treatment between the two groups but P-value for within-subject effect was significant (< 0.001); i.e. reduction in total BPRS scale at the end of 1-week compared with the next week demonstrated improvement in the symptoms until the end of the study.

Our finding was unlike the remission that was seen after 6 sessions of right unilateral ECT in a case report among persistent METH psychosis from Massachusetts. The mechanism through which ECT might benefit METH psychosis is still speculative. However, it should be noted that striatal dopamine is reduced in rhesus monkeys[30] and rodents[31,32] exposed to METH along with the postmortem tissues from people with METH addiction.[33] Conversely, chronic ECS increases striatal dopamine in healthy rats.[34,35] Neuroimaging findings in chronic METH users have demonstrated a decrease in dopamine transporters that recovers only gradually after prolonged abstinence, suggesting dopamine terminal damage which is very slow to repair.[36] Recent animal data have recommended that chronic METH exposure leads to decreased immediate early gene (e.g., c-fos) expression.[37]

In contrast, ECS is associated with increased brain-derived neurotropic factor, nerve growth factor, and immediate early gene (e.g., c-fos) expression; therefore, ECT may confer neuroprotection and neuroplasticity.[35] Thus, it can be speculated that ECT counteracts the effects of METH, at least partially through normalization of the neural environment or by stimulating the proliferation of nerve terminals. However, extrapolation from animal studies must be cautiously done, since such studies may use larger amphetamine doses than those used by humans and may not mimic the gradual escalation of dosage typical in human abusers, which is a pattern that may attenuate amphetamine-associated neurotoxic effects.[32]

Results of this investigation revealed that few sessions of ECT in persistent METH psychosis will not lead to remission in all patients. Furthermore, it is not clear if increasing ECT sessions may lead to remission, at least in a greater number of patients,

or not, because it is speculated that ECT counteracts the effects of METH, at least partially, through normalization of the neural environment or by stimulating the proliferation of nerve terminals, which may require more ECT sessions. It is also essential to conduct studies with a large enough sample size and increase the number of ECT sessions to evaluate remission rate after ECT in persistent METH psychosis, because safety, efficacy, and effectiveness cannot be evaluated in pilot studies.[26]

Conclusion

This research demonstrated that few sessions of ECT in persistent METH psychosis will not lead to remission in all patients. Therefore, it is essential to conduct further investigations with a large enough sample size and increase the number of ECT sessions to evaluate the effectiveness of ECT because safety, efficacy, and effectiveness cannot be evaluated in pilot studies; and increase the number of ECT sessions in order to it is not clear if increasing ECT sessions may lead to remission or not.

Conflict of Interests

The Authors have no conflict of interest.

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اثربخشی الکتروشوک در درمان سایکوز مقاوم مت‌آمفتامین: یک مطالعه مقدماتی

دکتر حسن ضیاءالدینی[۱]، دکتر تکتم روحبخش[۲]، دکتر نوذر نخعی[۳]، دکتر علیرضا غفاری نژاد[۳]

مقاله پژوهشی

چکیده

مقدمه: سایکوز پایدار مت‌آمفتامین سایکوزی است که بیش از یک ماه پس از قطع مصرف مت‌آمفتامین ادامه یابد و درمان مؤثری برای آن وجود نداشته باشد. هدف از انجام این مطالعه، بررسی اثربخشی الکتروشوک در بیماران سایکوز پایدار مت‌آمفتامین بستری در بیمارستان شهید بهشتی کرمان از ۱۵ شهریور سال ۱۳۹۱ تا ۱۵ شهریور سال ۱۳۹۲ بود که این بیماران با الانزاپین (Olanzapine) بهبودی کاملی نداشتند.

روش‌ها: مطالعه حاضر یک مطالعه مقدماتی (Pilot study) در بیماران بستری در بیمارستان بود. پس از چهار هفته درمان با الانزاپین، از ۷۱ نفر بیمار مورد مطالعه، ۱۰ بیمار با وجود پاسخ به درمان، بهبودی کاملی در علایم سایکوتیک نداشتند. بر اساس جدول ارقام تصادفی، این ۱۰ بیمار به دو گروه تقسیم شدند که در ۵ بیمار درمان با الانزاپین تا دو هفته ادامه یافت و ۵ بیمار دیگر علاوه بر الانزاپین، ۶ جلسه الکتروشوک به صورت دو طرفه و یک روز در میان تا دو هفته دریافت کردند.

یافته‌ها: میزان پاسخ به الانزاپین در چهار هفته ابتدایی درمان ۷۸/۷ درصد بود. کاهش علایم بر اساس مقایسه نمره کل ارزیابی روان‌پزشکی مختصر (Brief psychiatric rating scale یا BPRS) در پایان هر هفته با هفته قبل تا پایان مطالعه مشاهده شد. اختلاف معنی‌داری در نمره BPRS بین دو گروه، در هفته چهارم و ششم وجود نداشت.

نتیجه‌گیری: این مطالعه مشخص کرد که جلسات محدود الکتروشوک در سایکوز پایدار مت‌آمفتامین در همه بیماران منجر به بهبود کامل سایکوز نمی‌شود.

واژگان کلیدی: سایکوز مت‌آمفتامین، درمان، درمان الکتروشوک

ارجاع: ضیاءالدینی حسن، روحبخش تکتم، نخعی نوذر، غفاری نژاد علیرضا. **اثربخشی الکتروشوک در درمان سایکوز مقاوم مت‌آمفتامین: یک مطالعه مقدماتی.** مجله اعتیاد و سلامت ۱۳۹۴؛ ۷ (۲-۱): ۲۳-۱۴.

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